Cognitive Behaviour Therapy for Posttraumatic Stress in Schizophrenia. A Randomised Controlled Trial

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Background. There is limited evidence for effective interventions in the treatment of posttraumatic stress symptoms within individuals diagnosed with schizophrenia. Clinicians have concerns about using exposure treatments with this patient group. The current trial was designed to evaluate a 16-session cognitive restructuring programme, without direct exposure, for the treatment of posttraumatic stress symptoms specifically within individuals diagnosed with schizophrenia.

Method. A multicentre randomized controlled single-blinded trial with assessments at 0 months, 6-months (post-treatment) and 12 months (follow-up) was conducted. Sixty-one participants diagnosed with schizophrenia and exhibiting posttraumatic stress symptoms were recruited. Those randomized to treatment were offered up to 16 sessions of cognitive behaviour therapy (CBT, including psychoeducation, breathing training and cognitive restructuring) over a 6-month period, with the control group offered routine clinical services. The main outcome was blind rating of posttraumatic stress symptoms using the Clinician Administered PTSD Scale for Schizophrenia (CAPS-S). Secondary outcomes were psychotic symptoms as measured by the Positive and Negative Symptom Scale (PANSS) and the Psychotic Symptom Rating Scale (PSYRATS).

Results. Both the treatment and control groups experienced a significant decrease in posttraumatic stress symptoms over time but there was no effect of the addition of CBT on either the primary or secondary outcomes.

Conclusions. The current trial did not demonstrate any effect in favour of CBT. Cognitive restructuring programmes may require further adaptation to promote emotional processing of traumatic memories within people diagnosed with a psychotic disorder.

Key words: Posttraumatic Stress, schizophrenia, cognitive-behavioural therapy, cognitive restructuring, randomised controlled trial, treatment.

Introduction

Individuals diagnosed with a psychotic disorder report suffering a high prevalence of stressful and traumatic life events (Bebbington *et al*. 2004) with childhood trauma specifically indicated as a risk factor for diagnosis (Matheson et al., 2013). The prevalence of posttraumatic stress disorder (PTSD) has been estimated to be aproximately15% within this group (Achim *et al*. 2011; de Bont *et al.*, 2015; Grubaugh *et al*., 2011). This co-morbid presentation is associated with a poor prognosis and increased use of healthcare (Switzer *et al*. 1999), contributing to calls for the routine assessment of trauma symptoms within the psychiatric system (Rose *et al*., 2012).

An increased awareness of the association between stressful life events and the onset and maintenance of psychotic symptoms has also contributed to the development of theoretical models aimed at informing psychological interventions (e.g. Morrison *et al*. 2003; Read *et al*. 2014; Steel *et al.* 2005). One approach is to work towards a cognitive-behavioural formulation in

which reactions to early trauma are embedded within a developmental framework. Links are made between these life events and psychotic experiences such as paranoia. On the basis of this formulation, interventions are adopted which target unhelpful appraisals driven by core beliefs, such as 'others are not to be trusted' (e.g. Smith *et al*. 2006).

An alternative approach is to adopt a psychological intervention that has been established as a treatment for the symptoms of posttraumatic stress within a non-psychotic patient population. One such established treatment is cognitive-behavioural therapy for PTSD (Foa, 2008) which includes exposure therapy and/or cognitive restructuring. Prolonged exposure to the memory of traumatic events in order to elaborate and contextualise these memories is a critical component of several evidence-based treatments for PTSD (e.g. Clark & Ehlers, 2004; Foa *et al*., 2005). A recent clinical trial suggests that exposure based interventions are effective in individuals diagnosed with a psychotic disorder (van den Berg *et al*. 2015). However, the sensitivity to stress associated with this group (Lataster *et al*., 2013) has led to concern among clinicians about using this approach (Meyer *et al*. 2014). This is a challenge within the delivery of U.K. psychosis services, where only 10% of eligible service users receive any type of psychotherapy (The Schizophrenia Commission, 2012).

Clinical studies of participants diagnosed with PTSD but not psychotic disorder indicate that cognitive restructuring and exposure are equally effective in supporting emotional processing of trauma memory and reduction of PTSD symptoms (Marks, 1998; Resnick *et al*. 2003; Tarrier *et al*. 1999). Therefore, based on the assumption that cognitive restructuring is less stressful than exposure therapy, Mueser and colleagues developed a structured cognitive-behavioural program for treating PTSD in individuals exhibiting a range of complex presentations, including people diagnosed with schizophrenia, with a primary focus on cognitive restructuring (Mueser *et al*. 2008; Mueser *et al*. 2015). The 12-16-session program includes psychoeducation, breathing training, and cognitive restructuring and has been shown to be effective in reducing PTSD symptoms in those diagnosed with severe mental health problems in two randomized controlled

trial (Mueser et al., 2008; Mueser *et al*. 2015). However, these studies contained samples in which the majority of participants had severe mood disorders, and less than one-third had a schizophrenia-spectrum disorder. Therefore the program needs further investigation to determine its effectiveness for people diagnosed with a psychotic disorder, and schizophrenia or schizoaffective disorder in particular. This was the aim of the current study.

It was hypothesized that CBT in addition to treatment as usual would result in large reductions in posttraumatic stress symptoms.

Method

Trial Design

This was a single-blind randomised controlled trial of CBT vs treatment as usual with a 6-month treatment phase and a 12-month follow-up phase. Up to 16 sessions of treatment were available within the 6-month period, with 6 or more sessions being considered minimal exposure to the treatment protocol.

Robust procedures were adopted to minimize the risk of interviewers being able to identify the group allocation of participants. Blind was broken in 10 (6.4%) of the 157 completed assessments. Of these, 7 (4.5%) assessments were subsequently conducted by a new masked interviewer. Three (1.9%) of assessments took place with the mask broken and an audio recording was sent to a masked interviewer to conduct the ratings.

All interviewers were trained for reliability on the CAPS-S and PANSS, and subsequently attended monthly meetings in which a trial assessment was rated by all interviewers in order to assess for any drift in inter-rater agreements.

The study was conducted in two large NHS Trusts located in the South of England, namely Berkshire Healthcare Foundation Trust (BHFT) and North East London Foundation Trust

(NELFT). The trial was given ethical approval by Berkshire REC SC/09/H0505/85 and was registered as ISTCRN 67096137.

Sample-size calculation

Power analysis was based on obtaining a reduction of 15 points on the CAPS-S, which represents a clinically meaningful change (Weather et al., 2001). Using a conservative estimate of the standard deviation of the change score (SD=15) derived from a previous study (Rosenberg *et al*. 2004), and assuming that mean scores in the TAU group do not change over the treatment period, a mean reduction of 15 points in the CBT group translates into a large effect size (d=1). To detect such an effect, or a larger one, using an independent samples t-test at the conservative 5% significance level (two-tailed) with 95% power, a sample size of 26 per group would be needed. Assuming a 15% drop-out rate, a sample of 31 patients per group was required.

Participants

Participants were eligible if they were aged between 18 and 65, had stable living arrangements, met DSM-IV (American Psychiatric Association, 1994) criteria for schizophrenia, schizoaffective disorder or schizophreniform disorder, and met DSM-IV criteria for PTSD. Exclusion criteria were organic impairment or insufficient command of English.

Measures

Screening

History of trauma exposure was evaluated with the Trauma History Questionnaire (THQ; Green, 1996), which has been previously adapted for use with people with severe mental health problems (Mueser *et al*., 2008). The original 16-item version was extended to include two extra items relevant to the current population, namely psychiatric treatment and psychotic experiences which were experienced as a threat to the self (see Picken & Tarrier, 2011). Eligibility was initially

assessed using a brief self-report measure, the Posttraumatic Stress Disorder Checklist, Civilian version (PCL-C, (Blanchard *et al*., 1996)) which contains 17 items on a 5-point scale (range 17-85).

Primary Outcome

PTSD symptom severity was assessed with the Clinician Administered PTSD Scale for Schizophrenia (CAPS-S; Gearon *et al*, 2004). The CAPS-S is an adapted version of the CAPS (Blake *et al*. 1995) which has been shown to be reliable and valid when used with people diagnosed with severe mental health problems (Mueser *et al*. 2001). For each symptom, a frequency and intensity rating is provided, with overall severity scores computed by summing the frequency and intensity scores for all of the PTSD symptoms (CAPS–Total).

Secondary outcome

Positive symptoms of psychosis were assessed with the Positive and Negative Syndrome Scale (PANSS, Kay *et al.* 1987). The PANSS includes items measuring positive symptoms, negative symptoms, and other symptoms, and is used widely in research with people daignosed with schizophrenia..

Other measures

Severity of hallucinations and delusions were assessed using the Psychotic Symptom Rating Scale (PSYRATS, Haddock *et al.* 1999).

Trauma-related cognitions were assessed with the Posttraumatic Cognitions Inventory (PTCI, Foa *et al.* 1999), a self-report measure of common negative beliefs about oneself, other people, and the world, which relate to traumatic experiences. Greater endorsement of negative beliefs is indicated by a high score.

Depression and anxiety were rated with the Beck Depression Inventory (BDI-II; Beck *et al*. 1996) and the Beck Anxiety Inventory (BAI; Beck & Steer, 1990).

Functioning was assessed using the Global Assessment of Functioning (GAF; Hall, 1995) which produces a rating from 0 to 100, with a high score indicating higher functioning.

Quality of Life was measured using the Quality of Life (QLS) scale (Heinrichs *et al*. 1984).

Procedures

Recruitment was conducted by trained research assistants and clinical studies officers from the NIHR Clinical Research Network Mental Health at both sites. Recruitment was systematic with each mental health team responsible for a geographical location being approached in turn. Research assistants contacted all care coordinators in order to identify potentially eligible participants.

When individuals provided consent, research assistants then checked health care records to assess eligibility in relation to primary diagnosis and demographics. The modified THQ was then completed with potential participants identifying which event was currently associated with the most distress. The PCL-C was then completed in relation to that event. If an individual scored 44 or higher they were invited to complete a clinical interview to assess the symptoms of PTSD with the CAPS-S.

Recruitment commenced in May 2010 with an entry criterion of diagnostic PTSD. Due to lower than anticipated recruitment rates, in May 2011 the CAPS-S entry criterion was amended to include those who fulfilled criteria A (event), B (intrusive symptoms), E (time duration) and F (functional impairment) but not necessarily criterion C (avoidance) or D (hyperarousal). Traumatic events related to psychosis were not required to meet criterion A (objective physical threat) for eligibility, although a subjective perception of threat to self or others was present. A maximum of 4 separate traumas were assessed using the CAPS-S within any potential participant in order to determine eligibility.

Eligible participants were randomised immediately after the completion of baseline assessments. Block randomisation was conducted independently of the research team through the OpenCDMS database specifically developed for the study and was stratified for site and PTSD symptom severity (CAPS-S ≥ 65).

Interventions

CBT for PTSD. The intervention followed the protocol adopted in previous trials (see Mueser *et al*., 2008) with minor adaptations made for use in the U.K. The 12-16 session programme followed a structured format and included handouts, worksheets and homework assignments. All sessions were conducted at the clients' local community mental health centre or at their home. There was regular contact and coordination between trial therapists and NHS treatment teams. Individuals allocated to CBT also received the routine clinical services available to the TAU (Treatment as Usual) group.

TAU. Clients assigned to TAU continued to receive the usual services available to them through their clinical teams. Type and dose of medication was determined by NHS clinicians, and was not affected by the trial protocol.

Trial Therapists

There were thirteen trial therapists comprised of 7 clinical psychologists, 3 BABCP accredited CBT therapists, 2 counselling psychologists and 1 trainee clinical psychologist. Of the 30 cases allocated to treatment, 23 (77%) were seen by clinicians with over 5 years experience of delivering psychological interventions with individuals diagnosed with psychosis, 3 (10%) by clinicians with one to five years experience and 4 (13%) by clinicians with less than one years experience. Training in the trial protocol was delivered by JG over a 2-day period. Ongoing fortnightly supervision was delivered by BS and AH for all therapists.

Overall, 25% of recorded sessions were assessed using a measure of competence and adherence and were judged to be of at least an adequate level.

Statistical Analysis

To allow for correlation between the post-treatment and follow-up value of the same variable, linear mixed (covariance pattern) models with unstructured covariance matrices were fitted using SAS PROC MIXED**.** Otherwise known as marginal models, this approach is equivalent to fitting models with random intercepts that vary at the level of study participants. Maximum likelihood estimation procedures ensure that inferences will remain valid in the presence of missing data, provided the missing value generating mechanism is missing at random (not missing completely at random) and is therefore a less restrictive approach than the conventional complete case analysis. Analyses were adjusted for baseline response, whether or not inclusion in the study was based on the initial eligibility criteria and for the randomisation stratifier (CAPS-S severity with a cut-off at 65) by including these as fixed explanatory variables in the models. Time and treatment-by-time interaction terms were retained in all models and group differences estimated at each post randomisation time point. Residual diagnostics were conducted to check violation of normality assumptions. Post treatment and follow up effect sizes based on adjusted treatment means, are computed using Cohen's *d* along with 95% confidence intervals. The same approach was adopted for the main outcome (CAPS-S) as for the secondary outcome (PANSS positive) and the other outcome measures.

Results

Sample characteristics

In total 1,465 referrals were provided for the trial, of whom 209 gave consent to be assessed and 61 were eligible. See Fig.1 for the trial CONSORT (Consolidated Standards of Reporting Trials) diagram. The characteristics of the participants allocated to CBT and TAU are summarized in

Table 1. The two trial arms were similar in terms of diagnostic, demographic and baselines measures.

The most commonly reported traumatic events associated with eligibility was the experience of a distressing psychotic episode (n=11, 18%) and sexual abuse whilst under the age of 16 (n=11, 18%). A range of other events were reported, with none being experienced by more than 10% of the sample.

For those allocated to the CBT group, the mean number of therapy sessions was 12.3 (range = 0-16, *SD*=5.3). Twenty-one participants (70%) received the full protocol of 12 sessions or more and 26 (87%) received at least 6 sessions, which is considered minimal exposure to the intervention (Mueser *et al*., 2015).

Outcome measures

There was no significant difference between the intervention and control group in the primary outcome of PTSD symptoms on the CAPS-S at the end of treatment ($p>0.1$, between-group d = -0.26 (-0.84, 0.32)), or in the secondary outcome of the positive symptoms on PANSS positive ($p>0.1$, between-group d = 0.32 (-0.26, 0.91)). There were no significant differences between groups within any of the other outcome measures (see Table 2).

Although there was no significant difference between the treatment and control groups regarding the main outcome of PTSD symptoms, the severity of the posttraumatic stress symptoms declined within the combined group over the 12-month period (CBT: F=4.41; $p<0.01$; TAU: F=8.51; $p<0.01$).

Conclusion

The current study is one of only two published clinical trials that have aimed to evaluate the effectiveness of an evidence-based psychological intervention for PTSD solely within individuals

diagnosed with a psychotic disorder. Our results do not provide evidence for cognitive restructuring with no direct exposure, the protocol adopted in the current study, reducing the symptoms of PTSD or psychosis beyond that achieved through treatment as usual within this group. Eighty-seven percent of individuals allocated to treatment received a 'dose' of the protocol (i.e. at least 6 sessions). Treatment delivery data combined with the high level of methodological rigour as regards blind assessments, randomisation procedures and data analysis suggests that the non-significant outcome is a valid result. The confidence intervals of the effect size suggest that there is less than a 5% chance of the 'true' effect size being above 0.32. This result should be considered within the context of meta-analyses which suggest the overall effect size of generic CBT for psychosis to be in the region of 0.4 (Wykes *et al.* 2008; van der Gaag et al. 2014).

Given that Mueser et al. (2008, 2015) report that a cognitive restructuring intervention is effective for a heterogeneous sample suffering from a range of severe mental health problems, it is important to consider any methodological differences between these trials and the current study.

First, unlike Mueser et al. (2008, 2015), the current study included individuals suffering from posttraumatic stress symptoms but who did not exhibit a fully diagnostic level of PTSD. Therefore, further analyses were conducted excluding the participants within the current sample who did not present with a diagnostic level of PTSD symptoms. There was no effect of treatment within this fully diagnostic subgroup (see Table 2). Although the small sample size limits the reliability of this post-hoc analyses, the result is in line with a recent meta-analysis which indicates that the Mueser et al (2008, 2015) studies did not produce a significant treatment effect within the subgroup of their samples which were diagnosed with schizophrenia (Sin & Spain, 2016). However, it should be noted that the majority of participants in the Mueser et al. studies were diagnosed with severe levels of PTSD (i.e. a CAP-S score above 65), whilst the current study did not recruit enough participants in this subgroup in order to conduct meaningful analysis.

Therefore, our study does not exclude the possibility of the cognitive restructuring programme being effective with this more severe group.

Second, the current study included psychotic experiences and psychiatric treatment as potentially eligible stressful life events whereas Mueser et al (2008, 2015) did not. Eleven participants were eligible on the basis of their reaction to a past psychotic episode although none were eligible in relation to psychiatric treatment. Whilst the concept of post-psychotic PTSD has gained validity (Mueser *et al*., 2010), specific traumatic events may be associated with distinct responses to a psychological intervention. However, further post-hoc analyses in which these eleven participants were excluded revealed a non-significant treatment effect (see Table 2).

Both the intervention and control groups showed a significant reduction in PTSD symptoms over the 12-month study period. Van den Berg *et al*., (2015) also report a reduction (16 points) in the CAPS ratings of individuals who did not receive a psychological treatment within a 12-month period. These results are inconsistent with data indicating that co-morbid PTSD is indicative of a poor prognosis within individuals diagnosed with a psychotic disorder (Switzer *et al*. 1999). One possibility is that individuals who consent for treatment within a clinical trial are a 'help-seeking' subgroup and may not be representative of a wider clinical group exhibiting the same symptom profile. Another possibility is that the symptoms of posttraumatic stress may fluctuate over time. Therefore, a portion of the current sample may have entered the clinical trial at a more severe stage of a cyclical presentation and would experience a subsequent drop in symptoms whether or not they received an intervention. It is of note that the test-retest reliability of the CAPS when used with people diagnosed with severe mental health problems increases in line with the severity of PTSD symptoms (Mueser et al, 2001), suggesting that symptom fluctuation is more likely to occur within the non-severe group which are the majority within the current study. This effect is likely to have contributed to the unexpected significant reduction of symptoms within the control group. It is therefore a limitation of the current trial that the stability

of posttraumatic stress symptoms were not assessed over a period of time before eligibility was confirmed.

Our aim was to evaluate the effectiveness of cognitive restructuring for the treatment of posttraumatic stress symptoms specifically within individuals diagnosed with schizophrenia. Our results do not provide evidence for the use of this intervention within this group. A recently published process analysis based on recordings of the intervention sessions from the current study suggests that, whilst engagement remained high, the level of emotional processing may not have been sufficient to support adaptation to trauma memories and reductions in PTSD symptoms (O'Driscoll et al., 2016). Therefore, cognitive restructuring programmes may require further adaptation to promote emotional processing of traumatic memories within people diagnosed with a psychotic disorder.

To conclude, the current results to not provide support for the use of a cognitive restructuring alone for the treatment of posttraumatic symptoms within people diagnosed with schizophrenia. However, given that the Mueser et al. (2015) study was targeted specifically at individuals suffering from severe levels of PTSD, it may be that such an approach is effective within the severe subgroup. However, any future research would benefit from an adapted protocol. Therefore, despite anxiety within some mental health professionals, current evidence indicates that exposure is required in order to treat trauma symptoms within this group. The positive symptoms of psychosis may be exacerbated by the presence of decontextualiased intrusive memories, such that trauma memories may need to be directly retrieved and elaborated in order for the symptoms of posttraumatic stress to reduce (Longden *et al*., 2012; Steel, 2015; Steel *et al*., 2005). This conclusion would be consistent with the positive results recently obtained using direct memory exposure interventions (Prolonged Exposure and EMDR) for the treatment of PTSD in psychotic disorder (van den Berg *et al*. 2015).

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Declaration of Interest

None.

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Figure 1. CONSORT diagram of recruitment to the study. TAU = treatment as usual; CBT = cognitive behavioural therapy.

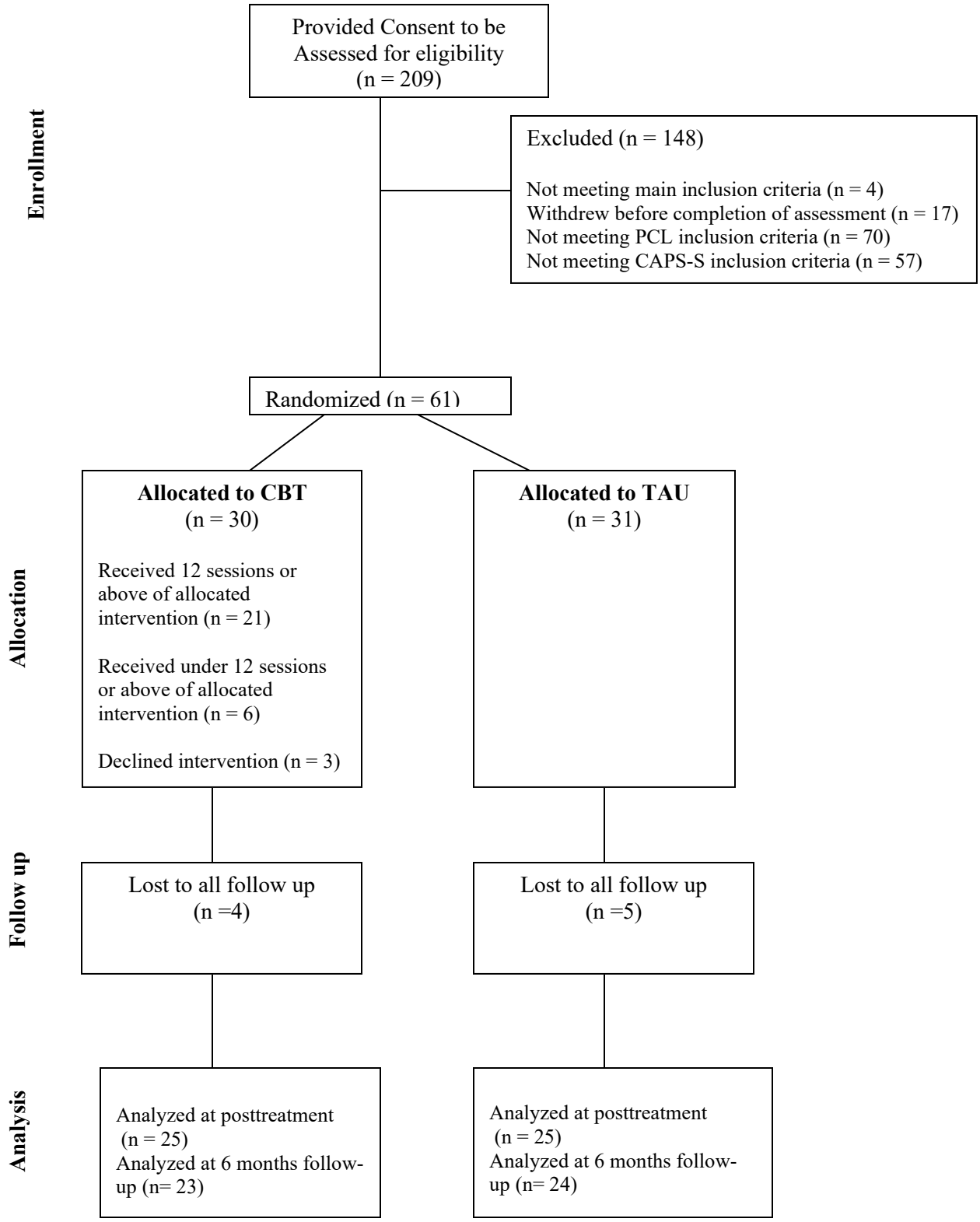

Table 1. *Baseline characteristics of the sample*

Variable	CBT (n=30)	TAU (n= 31)	Total (n=61)
Demographics			
Mean age in years (SD)	43.8 (10.1)	40.7 (10.2)	42.3 (10.2)
Male (%)	60.0	64.5	62.3
White (%)	74.2	70.0	72.1
Age left formal education	17.0 (2.3)	17.5 (4.5)	17.2 (3.6)
Currently Employed (%)	3.3	0.0	1.6
Primary Diagnosis			
Schizophrenia	66.7	80.6	73.8
Schizoaffective disorder	33.3	19.4	26.2
Psychiatric history*			
Prior psychiatric Hospitalization (%)	90.0	92.6	91.2
Mean number of prior Admissions	4.5 (3.7)	5.4 (10.1)	4.9 (7.4)
Mean age at first contact with mental health services	25.0 (12.1)	24.4 (10.5)	24.7 (11.3)

*Data on psychiatric history were missing for 4 patients in the CBT group.

Table 2 *Assessment of CBT and TAU groups during the intervention and follow-up periods*

Outcome	Group	Means (SD)			Group effect (6m)			Group effect (12m)		
		Base	6m	12m	*t*	*p*	*ES* (95% CIs)	*t*	*p*	*ES* (95% CIs)
CAPS-Total	CBT	49.9 (22.3)	41.7 (22.9)	34.4 (24.4)	0.88	0.39	-0.26 (-0.84,0.32)	0.86	0.39	-0.29 (-0.88,0.30)
	TAU	54.6 (20.0)	37.8 (25.9)	30.4 (24.0)						
CAPS-Total *Diagnosed PTSD group*	CBT	65.4 (17.5)	44.0 (22.4)	32.8 (26.5)	0.16	0.87	-0.06 (-0.82,0.69)	0.44	0.66	0.22 (-0.57,1.01)
	TAU	66.6 (14.1)	44.5 (27.9)	40.7 (26.8)						
CAPS-Total *Excluding Post-Psychotic PTSD*	CBT	50.1 (21.8)	42.6 (23.7)	36.8 (25.3)	0.67	0.51	-0.23 -0.89,0.42)	1.41	0.16	-0.55 (-1.23, 0.12)
	TAU	56.8 (20.2)	40.9 (26.6)	29.7 (24.0)						
PANSS pos	CBT	19.1 (6.0)	17.8 (5.6)	17.0 (5.6)	-1.3	0.20	0.32 (-0.26,0.91)	-0.82	0.416	0.24 (-0.37,0.84)
	TAU	18.3 (5.3)	19.8 (6.6)	18.4 (6.4)						
PANSS neg	CBT	16.3 (6.1)	15.0 (5.7)	15.0 (4.6)	-2.31	0.03	0.46 (-0.13,1.05)	-0.88	0.382	0.21 (-0.40,0.81)
	TAU	15.3 (5.4)	16.4 (5.4)	16.1 (4.5)						
PSYRATS Hall	CBT	16.9 (15.0)	16.8 (13.4)	13.7 (13.8)	1.36	0.18	-0.26 (-0.84,0.32)	0.31	0.755	-0.08 (-0.70,0.54)
	TAU	16.4 (13.7)	14.0 (13.2)	14.0 (14.8)						
PSYRATS Del	CBT	11.8 (8.0)	10.0 (8.5)	8.5 (8.0)	0.53	0.60	-0.14 (-0.72,0.44)	-0.19	0.849	0.05 (-0.56,0.67)
	TAU	12.5 (7.3)	10.7 (7.5)	10.8 (7.6)						

BDI	CBT	30.3 (10.5)	24.3 (14.2)	21.9 (11.3)	-0.31	0.76	0.14 (-0.53,0.81)	0.06	0.950	-0.03 (-0.67,0.62)
	TAU	23.0 (10.2)	21.4 (11.1)	18.6 (11.3)						
BAI	CBT	26.9 (12.6)	21.8 (15.2)	19.4 (14.6)	-0.76	0.45	0.29 (-0.39,0.98)	-1.02	0.318	0.43 (-0.21,1.07)
	TAU	21.3 (10.2)	19.8 (11.9)	22.4 (15.6)						
PTCI	CBT	159.2 (43.8)	140.1 (42.9)	127.7 (49.7)	0.09	0.92	-0.04 (-0.71,0.63)	-0.42	0.677	0.18 (-0.47,0.82)
	TAU	162.8 (29.7)	142.3 (49.8)	132.5 (47.2)						
QLS	CBT	25.0 (7.6)	23.0 (9.8)	25.4 (7.2)	-1.04	0.31	0.40 (-0.26,1.06)	-0.81	0.426	0.24 (-0.41,0.88)
	TAU	26.4 (6.2)	26.0 (6.4)	26.0 (5.0)						
GAF	CBT	55.9 (11.0)	61.6 (10.0)	61.3 (9.8)	0.43	0.67	0.09 (-0.51,0.70)	0.69	0.495	0.18
	TAU	56.6 (12.0)	60.8 (8.3)	58.6 (11.0)						(-0.41,0.77)

BDI n = 25,25 / 19,7 / 20,19
BAI n = 25,25, / 18,17 / 20,20
GAF n = 29,29 / 23,21/ 23,23
PTCI n = 26,27/18,18/20,19
QLS n = 25,25 / 19,19 / 20/19
Effect size (ES) measures change from baseline in CBT relative to change in TAU.